Diagnostic Ophthalmology
Ophtalmologie diagnostique

Bruce H. Grahn, Lynne S. Sandmeyer

History and clinical signs

A 4-year-old, female, Labrador retriever was referred for examination by the ophthalmology service at the Western College of Veterinary Medicine. The owner had noted nyctalopia, and some general visual impairment. The menace responses, and the palpebral, direct and consensual pupillary light, and oculocephalic reflexes were present in both eyes. Schirmer tear test (Schirmer Tear Test Strips; Alcon Canada, Mississauga, Ontario) values were within normal reference ranges in both eyes. The intraocular pressures were estimated with an applanation tonometer (Tonopen XL; Biorad Ophthalmic Division, Santa Clara, California, USA) and were 16 mmHg and 21 mmHg in the right and left eyes, respectively. The pupils were dilated with tropicamide (Mydriacyl; Alcon Canada, Mississauga, Ontario). Biomicroscopic examination (Osram 64222; Carl Zeiss Canada, Don Mills, Ontario) did not reveal any other anterior segment abnormalities. Examination of the posterior segments of both globes was completed with an indirect ophthalmoscope (Heine Omega 200; Heine Instruments Canada, Kitchener, Ontario). We photographed (Kowa RC2; Kowa, Japan) both fundi and the right fundus photograph is provided for your assessment (Figure 1).

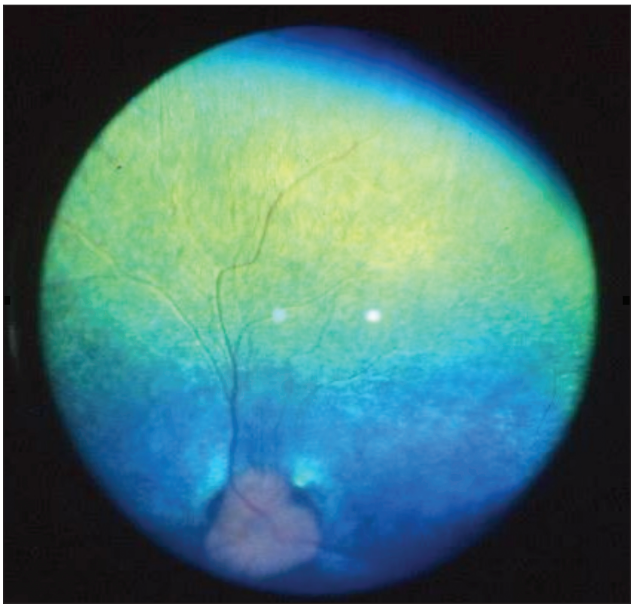

Figure 1. A photograph of the right fundus of a 4-year-old Labrador retriever. The left fundus was similar in appearance.

What are your diagnoses, differential diagnoses, and prognosis?

Our diagnosis was progressive retinal atrophy. Differential diagnoses that were considered were chronic sudden acquired retinal degeneration (SARD), and other acquired retinal degeneration (toxins, etc). These were excluded by the lack of rapid onset of blindness that is pathgnomonic for SARD, and a lack of exposure of the dog to potential toxins. The prognosis for vision is poor, and we recommended that the eyes be re-examined by an ophthalmologist on a yearly basis to monitor the development of cataracts and lens-induced uveitis.

Progressive retinal atrophy is an inherited retinal degeneration that may manifest in any purebred dog. This is a diverse group of inherited disorders with many mutations identified, and several yet to be discovered (1). The age of onset varies with the type of retinal degeneration diagnosed. Early onset progressive retinal atrophy may manifest in the first months of life, while later onset progressive retinal atrophy will develop signs of nyctalopia when the dog is a few years old (1).

It is appropriate to classify retinal atrophy as a dysplasia or degeneration. Photoreceptor dysplasia manifests with abnormalities in the photoreceptor that develop prior to maturation of the retina (the affected dog is $<$ 8 weeks old). Photoreceptor degenerations manifest with outer segment degeneration and are detected after the retina is fully mature (the dog is $>$ 8 weeks old). The clinical manifestations of these syndromes usually include nyctalopia, and all progress to complete blindness gradually. Photoreceptor degenerations render most affected dogs blind by 7 years of age.

The dog herein is appropriately diagnosed as having a progressive retinal atrophy, which is an inherited retinal degeneration. The retina will progressively atrophy over the next 1 to 2 years until the dog is completely blind. The ophthalmoscopic manifestations of progressive retinal atrophy include atrophy of the retinal vasculature, retinal thinning and tapetal hyperreflectivitiy, and pigment clumping within the retinal pigment epithelium as illustrated in Figure 1. These manifestations are usually identifiable by indirect ophthalmoscopic examination in dogs 2 to 3 years of age (1). The diagnosis can be confirmed much earlier (6 months to 2 years of age) by using scotopic and

Department of Small Animal Clinical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan S7N 5B4.

photopic electroretinography (2). Some of the mutations have been identified in dogs (1,3) and this has allowed mutational testing on harvested DNA. Cheek swabs and blood samples are routinely submitted for confirmation of the diagnosis and identification of carriers and selection of breeding dogs in populations where the disease is evident.

The prognosis for vision is poor, although gene therapy has restored vision in selected research dogs with some degenerative retinal disorders. Dogs with extensive degeneration, such as the Labrador retriever in this paper, will likely not benefit from such treatments as most of the retinal tissue is completely atrophic. Complete retinal atrophy will develop, and unfortunately the release of degenerative retinal by-products will induce cataract development. Cataracts usually cause phacolytic uveitis, zonular degeneration, and lens luxation; often, secondary glaucoma develops in these dogs. Therefore, we recommend yearly re-examinations by a veterinary ophthalmologist to allow for early diagnosis and medical or surgical management of these common often initially subclinical sequelae.

References

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